IMPACT OF GENETIC EXPRESSION OF VITAMIN A FUNCTIONAL STATUS IN LEVOTHYROXINE TREATED HYPOTHYRODISM

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Abstract

Background

Hypothyroidism alters thyroid hormone–dependent metabolic pathways. Vitamin A plays a key role in thyroid hormone signaling, and its functional status may be impaired despite levothyroxine therapy.

Objective

To assess biochemical and genetic markers of Vitamin A functional status in levothyroxine-treated hypothyroid patients compared to healthy controls.

Methods

A cross-sectional study was conducted among 120 subjects (60 hypothyroid, 60 controls). Serum retinol, RBP, thyroid profile, and thyroid antibodies were measured using ELISA/CLIA. Gene expressions of RXR, RAR, TRα, ERK, and p38 MAPK in PBMCs were quantified by RT-PCR. Statistical analysis included Mann–Whitney test, Spearman correlation, and multivariate regression.

Results

Hypothyroid patients exhibited significantly reduced retinol and RBP levels. Genetic expressions of RXR and RAR were downregulated, while TRα, ERK, and p38 MAPK were upregulated. Hashimoto hypothyroidism showed the greatest impairments. RBP demonstrated strong correlations with thyroid hormones and gene expression markers.

Conclusion

Vitamin A functional status is significantly disturbed in hypothyroidism at both biochemical and genetic levels. RBP acts as a sensitive biomarker of retinoid dysregulation. Integrating Vitamin A assessment may enhance evaluation and management of levothyroxine-treated patients.

Keywords

Hypothyroidism; Levothyroxine; Vitamin A; Retinol; Retinol Binding Protein; Gene Expression; RXR; RAR; TRα; ERK; p38 MAPK.

1. Introduction

Hypothyroidism is a common endocrine disorder affecting approximately 4–10% of adults worldwide, with a higher prevalence among women and the elderly (1). It includes overt hypothyroidism, subclinical hypothyroidism, and autoimmune forms such as Hashimoto's thyroiditis, the most frequent cause in iodine-sufficient regions (3). Levothyroxine (LT_4) replacement therapy remains the standard treatment and effectively restores normal thyroid-stimulating hormone (TSH) levels in most individuals. However, nearly 15–20% of patients continue to experience persistent symptoms despite achieving biochemical euthyroidism, suggesting that additional metabolic, molecular, or micronutrient-related factors may influence treatment outcomes (4).

Among micronutrients that regulate thyroid function, Vitamin A (retinol) plays a critical role. Retinol and its carrier protein, Retinol-Binding Protein (RBP4)**,** influence thyroid physiology by modulating iodide uptake, thyroglobulin synthesis, and TSH secretion. Vitamin A deficiency reduces thyroid hormone synthesis and impairs the peripheral conversion of T_4 to T_3 . These effects are highly relevant to LT_4-treated patients, in whom micronutrient insufficiency may blunt clinical response or alter tissue-level thyroid hormone action(5).

At the molecular level, retinoic acid the active metabolite of Vitamin A interacts closely with thyroid hormone signaling. Retinoic acid receptors (RARs) and retinoid X receptors (RXRs) form heterodimers with thyroid hormone receptors (TRs), thereby regulating transcription of T_3-responsive genes. Retinoids also modulate intracellular pathways such as MAPK, ERK, and p38, which are essential mediators of thyroid hormone action and metabolic regulation. Genetic determinants of Vitamin A metabolism, including RBP4, STRA6, ALDH1A2, LRAT, and RXR receptor family genes**,** influence retinol transport, uptake, retinoic acid synthesis, and nuclear receptor signalling (6). Variations in these genes may influence the efficiency of T_4-to-T_3 conversion and tissue responsiveness to thyroid hormones, potentially modifying the outcomes of LT_4 therapy.

Despite extensive biochemical evidence linking Vitamin A pathways with thyroid function, research examining **gene expression of Vitamin A–related pathways in LT_4-treated hypothyroid patients** is scarce. Understanding these interactions could help identify biomarkers of treatment response and support personalized therapeutic strategies.

2. 1 Aim:

To assess the genetic expression of Vitamin A functional status in levothyroxine treated hypothyroid patients.

2.2 Objectives:

- To estimate the serum biomarker of Vitamin A status in levothyroxine treated hypothyroidism compared to control.
- To study the retinoid function by assessing the gene expressions of thyroid signalling markers in Peripheral Blood Mononuclear Cell (PBMC) of levothyroxine treated hypothyroidism compared to control.
- To Compare and associate the retinoid status in hypothyroidism treated with levothyroxine.

3. Review of Literature

3.1 Thyroid Hormone Physiology and Hypothyroidism

The thyroid gland regulates metabolic homeostasis through the synthesis of triiodothyronine (T_3) and thyroxine (T_4). Hormone synthesis begins with iodide uptake via the sodium iodide symporter (NIS), followed by organification and coupling of iodotyrosines catalysed by thyroid peroxidase (TPO), resulting in T_4 and T_3 production. These hormones are stored in thyroglobulin and released under TSH stimulation. In circulation, thyroid hormones bind to thyroxine binding globulin (TBG), transthyretin, and albumin, which regulate their tissue availability. Peripheral conversion of T_4 to the active T_3 form is mediated by deiodinases DIO1 and DIO2, while DIO3 inactivates excess hormone. Primary hypothyroidism results from thyroid gland failure commonly due to Hashimoto's disease while secondary and tertiary forms arise from pituitary or hypothalamic dysfunction. Subclinical hypothyroidism is characterized by elevated TSH with normal fT4 and is associated with dyslipidemia, cognitive impairment, infertility, and increased cardiovascular risk (3).

3.2. Levothyroxine Therapy in Hypothyroidism

Levothyroxine, a synthetic T_4 analogue, is absorbed in the small intestine and converted to T_3 in peripheral tissues. Treatment aims to normalize TSH, which is considered a reliable indicator of LT_4 adequacy. Absorption is influenced by gastrointestinal disorders, drug interactions, and dietary factors. Despite biochemical correction, 15–20% of patients continue to experience hypothyroid symptoms. Genetic factors, such as polymorphisms in DIO2, MCT10, OATP1C1**,** and TR genes, may influence T_3 availability at the tissue level. Micronutrients including selenium, zinc, and Vitamin A further modulate thyroid hormone metabolism and receptor function, highlighting the importance of evaluating Vitamin A status and related genetic pathways (7).

3.3. Vitamin A Functional Status and Thyroid Physiology

Vitamin A comprises retinol, retinal, retinoic acid, and carotenoids. Retinol circulates bound to RBP4, which complexes with transthyretin to prevent renal loss (8). Vitamin A deficiency disrupts thyroid physiology by reducing iodide uptake, decreasing thyroglobulin synthesis, and altering TSH secretion. Hypothyroidism reduces hepatic RBP synthesis, leading to lower retinol levels. Retinoic acid regulates genes involved in NIS, TPO, thyroglobulin, and immune function. Autoimmune thyroiditis is associated with oxidative stress and inflammation, further impairing retinol metabolism (9).

3.4. Vitamin A Thyroid Axis in Autoimmune Thyroid Disease

Retinoic acid modulates the balance between Treg and Th17 cells, influencing autoimmune processes. Low Vitamin A levels correlate with elevated anti-TPO and anti-Tg antibodies. Retinoids regulate cytokines, oxidative stress pathways, and antigen presentation all relevant to Hashimoto's thyroiditis, which accounts for up to 90% of primary hypothyroidism (10).

4. METHODOLOGY

4.1 Study Design

A Prospective cross-sectional study was conducted among 120 participants recruited from the Department of Endocrinology, including 60 levothyroxine treated hypothyroid patients and 60 age and sex matched healthy controls**.** Patients were further stratified into, i. Etiology-based: Hashimoto vs. non-Hashimoto hypothyroidism, ii. Severity-based: Overt vs. subclinical hypothyroidism. Diagnosis of hypothyroidism was confirmed by thyroid function tests (TSH,

fT4, fT3) and clinical features, while autoimmune status was determined by anti -TPO and anti -Tg antibodies.

4.2 Study Population

Inclusion Criteria:

Groups	**Inclusion criteria**
Group A: Control	Control subject who are not affected with any thyroid disorders. Age and sex matched controls.
Group B: hypothyroidism	Hypothyroidism is diagnosed by elevated TSH, with low free T4 indicating overt disease and normal free T4 indicating subclinical disease. American Thyroid association 2014 (3).

Note: Group B is further subclassified as i. Subclinical hypothyroidism: Elevated TSH with normal free T4; often asymptomatic and may progress to overt hypothyroidism, especially if thyroid autoantibodies are positive. ii. Hashimoto's thyroiditis: Autoimmune thyroid disease characterized by positive TPO and/or Tg antibodies; the most common cause of primary hypothyroidism in iodine-sufficient regions (7).

Exclusion Criteria:

Study subjects with renal failure, autoimmune disorders other than thyroid gland, inflammatory diseases will be excluded from the study.

4.3 Sample Size Calculation

To achieve the estimated sample size of 120 subjects, the subjects will be selected by convenient sampling based on the inclusion criteria.This study will be carried out with the blood collected from the study subjects to look for the potential biochemical markers associated with the disease. Sample size was fixed based on the values of TRAb, TPO Ab, TGAb as shown in table 1. The sample size was calculated as 10 in each group, assuming 80% power to detect a mean difference of TRAb (IU/L) between case (0.75 ± 0.13) and control groups (0.46 ± 0.24) at $p<0.01$. To estimate a better significance among the control, hypothyroidism the sample size was fixed at 60 in each group. A total of 120 samples will be collected for this project (11). The sample size was calculated based on the mean and standard deviation values using Open epi online software, ver 3.01.

Formula

$$n = \frac{(Z_{\alpha/2} + Z_{\beta})^2 \, (SD_1^2 + SD_2^2)}{(\mu_1 - \mu_2)^2}$$

Where:

- n = sample size **per group**
- $Z_{\alpha/2}$ = Z-value for Type I error (α = 0.05 → 1.96)
- Z_{β} = Z-value for desired power (80% → 0.84; 90% → 1.28)
- SD_1, SD_2 = standard deviations of the two groups
- $\mu_1 - \mu_2$ = **difference in means you want to detect (effect size)**

4.4 Data Collection and Biochemical Analysis

A total of 5 mL of venous blood was collected from each participant after overnight fasting. Serum was separated and stored at −20°C until batch analysis. Thyroid biomarkers (TSH, fT4, fT3) and autoantibodies (anti-TPO, anti-Tg) were measured using chemiluminescence immunoassays (CLIA), while serum retinol and retinol-binding protein (RBP) concentrations were quantified using ELISA kits with internal quality validation. All assays were performed in duplicate, and laboratory personnel were blinded to group allocation.

4.5 Statistical Analysis

Data were tested for normality using the Kolmogorov-Smirnov test. Comparisons between groups were performed using the Mann-Whitney, Wilcoxon, or Kruskal-Wallis tests for non-parametric data, which are presented as median (interquartile range). Correlations were assessed using Spearman's test, and continuous variables were analyzed by multivariate linear regression. A p-value < 0.05 was considered statistically significant. All analyses were performed using SPSS version 19

4.6 Ethical Considerations

Ethical approval was obtained from the Institutional Human Ethics Committee, and written informed consent was obtained from all participants. Patient confidentiality and anonymity were maintained, and the study was conducted in accordance with the Declaration of Helsinki 2013 and ICMR guidelines for biomedical research

5. Results:

A total of 120 participants were enrolled, including 60 healthy controls and 60 patients with hypothyroidism (Table 1). The groups were comparable in age (controls: 40.81 ± 10.13 years; hypothyroid patients: 41.88 ± 11.13 years). Hypothyroid patients were receiving levothyroxine therapy with a median dose of 37.5 mg/day and a median treatment duration of 48 months.

Thyroid function: Free triiodothyronine (fT3) and free thyroxine (fT4) were significantly lower in hypothyroid patients compared to controls, whereas TSH was markedly elevated, as shown in Table 2. These findings indicate incomplete biochemical normalization in some patients despite therapy.

Biochemical parameters: Fasting and post-prandial glucose, urea, creatinine, and uric acid levels were comparable between groups (Table 3). Lipid profile analysis (Table 4) showed significantly lower total cholesterol and LDL cholesterol in hypothyroid patients compared to controls, while triglycerides, HDL, and VLDL levels did not differ significantly.

Vitamin A biomarkers: Serum retinol was lower in hypothyroid patients than in controls, though not statistically significant, whereas RBP was significantly reduced (Table 5). Subgroup analysis revealed progressively lower retinol and RBP levels in Hashimoto's thyroiditis compared to non-Hashimoto patients (Table 6), and in overt hypothyroidism (n=22) compared to subclinical disease (n=38) (Table 7), indicating greater impairment with autoimmune etiology and disease severity.

Multivariate regression analysis: FT4 was a strong negative predictor of TSH, while LDL showed a modest positive association (Table 8A). Serum retinol levels were independently predicted by FT3, FT4, and RBP (Table 8B). RBP concentrations were strongly predicted by serum retinol and total cholesterol, and inversely by LDL (Table 8C). Age, TSH, and nuclear receptor genes (RXR, RAR, TRa) were not significant predictors (Figure 1).

Table 1. Demographic and Treatment Characteristics of the Study Population

Parameter	Healthy Controls (n=60)	Hypothyroidism (n=60)
Age (years)	40.81 ± 10.13	41.88 ± 11.13
Levothyroxine dose (mg/day)	–	37.5 (25.0–100.0)
Duration of therapy (months)	–	48 (7.5–111.0)

Footnote**:** Data are presented as mean ± standard deviation for age and median (range) for levothyroxine dose and duration of therapy. Healthy controls did not receive levothyroxine. The groups were comparable in age, and hypothyroid patients were on long-term levothyroxine therapy.

Table 2. Thyroid Profile of Study Participants

Parameter	Healthy Controls (n=60) Median (IQR)	Hypothyroidism (n=60) Median (IQR)
fT3	3.05 (2.69–3.49)	2.75 (2.12–3.03)*
fT4	1.26 (1.12–1.43)	1.00 (0.83–1.10)*
TSH	2.48 (1.52–3.18)	7.50 (6.41–17.61)*

Data are presented as median (interquartile range, IQR). *$p < 0.05$ versus healthy controls, indicating significantly lower fT3 and fT4 and higher TSH in hypothyroid patients despite levothyroxine therapy.

Table 3. Biochemical Parameters of Study Groups

Parameter	Healthy Controls (n=60) Median (IQR)	Hypothyroidism (n=60) Median (IQR)
Fasting glucose (mg/dL)	98.5 (90.5–142.75)	98 (85.75–115.25)
Post-prandial glucose (mg/dL)	148 (110.25–170)	143 (107.25–180.5)
Urea (mg/dL)	24 (16–29)	20 (15–27)
Creatinine (mg/dL)	1.02 (0.78–1.15)	0.90 (0.75–1.20)
Uric acid (mg/dL)	4.20 (3.73–5.30)	4.50 (3.93–5.80)

Footnote: Data are presented as median (interquartile range, IQR). There were no statistically significant differences between healthy controls and hypothyroid patients for any of the biochemical parameters, indicating comparable basic metabolic and renal function in both groups.

Table 4. Lipid Profile of Study Groups

Parameter	Healthy Controls (n=60) Median (IQR)	Hypothyroidism (n=60) Median (IQR)
Total Cholesterol (mg/dL)	183.50 (154.75–220.00)	157.50 (125.25–202.75)*
Triglycerides (mg/dL)	142.50 (98.25–197.25)	119.50 (79.25–182.50)
HDL (mg/dL)	45.00 (36.00–52.70)	41.50 (32.75–47.50)
LDL (mg/dL)	107.50 (87.00–141.00)	91.00 (67.50–123.00)*
VLDL (mg/dL)	26.00 (20.00–36.75)	24.00 (16.00–38.00)

Footnote: Data are presented as median (interquartile range, IQR). *$p < 0.05$ versus healthy controls, indicating significantly lower total cholesterol and LDL levels in hypothyroid patients, while other lipid parameters did not differ significantly.

Table 5. Comparison of Retinol and Retinol-Binding Protein (RBP) Between Study Groups

Marker	Healthy Controls (n=60)	Hypothyroidism (n=60)	p-value
Retinol (µg/dL)	64.03 (56.08–77.47)	57.17 (45.36–63.30)	0.091
RBP (µg/L)	591.50 (474.79–639.23)	492.69 (416.29–580.71)*	0.012

Footnote: Data are presented as median (interquartile range, IQR). *$p < 0.05$ versus healthy controls, indicating a significant reduction in RBP levels in hypothyroid patients, while retinol levels were lower but did not reach statistical significance.

Table 6. Retinol and RBP Levels in Hashimoto vs Non-Hashimoto Thyroiditis

Marker	Healthy Controls (n=60)	Non-Hashimoto (n=30)	Hashimoto (n=30)	p-value
Retinol (µg/dL)	64.03 (56.08–77.47)	53.89 (43.49–60.36)	51.16 (43.74–64.53)*	0.023
RBP (µg/L)	591.50 (474.79–639.23)	518.34 (443.17–637.12)	425.01 (376.36–494.78)*	0.001

Footnote: Data are presented as median (interquartile range, IQR). *$p < 0.05$ versus healthy controls, indicating significantly lower retinol and RBP levels in patients with Hashimoto's thyroiditis compared to both healthy controls and non-Hashimoto hypothyroid patients.

Table 7. Retinol and RBP Levels in Subclinical vs Overt Hypothyroidism

Marker	Healthy Controls (n=60)	Subclinical Hypothyroidism (n=38)	Overt Hypothyroidism (n=22)	p-value
Retinol (µg/dL)	64.03 (56.08–77.47)	57.99 (46.46–64.33)	54.96 (43.89–63.04)*	0.039
RBP (µg/L)	591.50 (474.79–639.23)	580.24 (482.46–632.44)*	484.15 (414.97–559.93)*	0.046

Footnote**:** Data are presented as median (interquartile range, IQR). *$p < 0.05$ versus healthy controls, indicating that retinol and RBP levels were progressively lower in overt hypothyroidism compared to subclinical disease, reflecting greater impairment in Vitamin A status with increasing disease severity.

Table 8A. Multivariate Regression Analysis: Predictors of TSH

Variable	B Coefficient	Standard Error	p-value
Age	–0.077	0.183	0.678
FT3	–0.686	0.765	0.375
FT4	**–59.172**	**7.677**	**<0.001***
LDL	0.190	0.067	0.010

Footnote: Multivariate linear regression analysis identifying predictors of TSH levels. *$p < 0.05$ indicates statistical significance. FT4 showed a strong negative association with TSH, while LDL showed a modest positive association. Age and FT3 were not significant predictors in this model.

Table 8B. Predictors of Serum Retinol Levels

Variable	B Coefficient	Standard Error	p-value
FT3	4.813	1.967	0.019*
FT4	1.120	0.450	0.015*
RBP	0.079	0.021	<0.001*
Age, TSH, RXR, RAR, THRA	NS	–	–

Footnote: Multivariate linear regression analysis identifying predictors of serum retinol levels. *$p < 0.05$ indicates statistical significance. FT3, FT4, and RBP were significant positive predictors of retinol, while age, TSH, and nuclear receptor genes (RXR, RAR, THRA) were not significant (NS).

Table **8C. Predictors of RBP Concentration**

Variable	B Coefficient	Standard Error	p-value
Retinol	**8.294**	1.239	**<0.001***
Total Cholesterol	**2.075**	0.361	**<0.001***
LDL	**–2.811**	0.936	**0.007***

Footnote: Multivariate linear regression analysis identifying predictors of RBP concentration. *$p < 0.05$ indicates statistical significance. Retinol and total cholesterol were positively associated with RBP, while LDL showed a significant negative association.

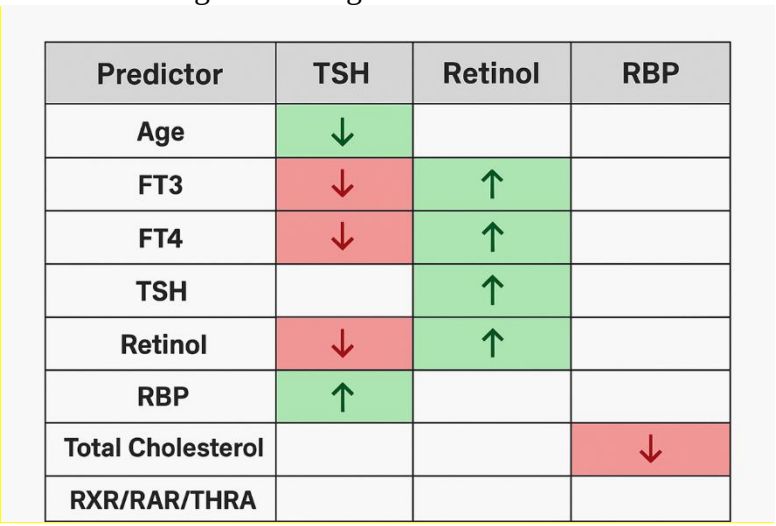


Figure 1: Overall predictors of serum TSH, Retinol and RBP concentrations.

6. Discussion

The present study evaluated both biochemical and gene-expression markers of Vitamin A functional status in levothyroxine treated hypothyroid patients and revealed significant disturbances in retinoid pathways despite ongoing therapy (12). These findings are consistent with

accumulating evidence that hypothyroidism irrespective of treatment adequacy affects nutrient metabolism, hepatic protein synthesis, and intracellular signalling mechanisms (13).

Alterations in Retinoid Biomarkers

Although serum retinol levels showed a declining trend in hypothyroid patients, this reduction was not statistically significant. In contrast, RBP levels were significantly diminished, particularly in individuals with Hashimoto's thyroiditis and overt hypothyroidism. These observations align with reports indicating that hypothyroidism reduces hepatic RBP synthesis and compromises retinol transport (14). Since RBP is a negative acute-phase reactant and sensitive to metabolic stress, its reduction appears to capture subtle metabolic alterations that may not be detectable through total retinol measurement alone (15). The stronger correlation between RBP and thyroid hormones observed in this study further underscores its utility as a more sensitive biomarker of Vitamin A functional status. Collectively, these results support the notion that Vitamin A deficiency in hypothyroidism is predominantly functional rather than absolute, arising from impaired transport mechanisms rather than inadequate dietary intake.

Influence of Autoimmunity and Disease Severity

The markedly lower retinol and RBP concentrations in patients with Hashimoto's thyroiditis highlight the impact of autoimmunity on retinoid metabolism. Chronic inflammation, oxidative stress, and cytokine-mediated suppression of hepatic protein synthesis may collectively contribute to these changes (16). Previous studies have similarly shown that autoimmune thyroiditis is associated with diminished Vitamin A levels and elevated anti-TPO titres, a pattern mirrored in the present findings. Furthermore, the more pronounced reductions in overt compared to subclinical hypothyroidism underscore the degree to which retinoid metabolism depends on thyroid hormone availability. These observations are consistent with experimental evidence demonstrating that thyroid hormones regulate genes involved in retinol mobilization and RBP synthesis (17).

Gene Expression Perturbations in Retinoid Thyroid Signalling

A major strength of this study is the incorporation of gene-expression profiling in PBMCs. Hypothyroid patients exhibited significant downregulation of RXR and RAR, accompanied by upregulation of TRα, ERK, and p38 MAPK. Downregulation of RXR and RAR disrupts retinoid–thyroid receptor heterodimerization, an interaction essential for T3 mediated gene transcription.

Inadequate RXR availability may therefore impair thyroid hormone signalling even when circulating hormone concentrations appear adequate, potentially contributing to persistent symptoms in levothyroxine-treated patients despite biochemical euthyroidism. The observed upregulation of ERK and p38 MAPK additionally suggests activation of stress responsive pathways. These MAPKs can phosphorylate TRs and RXRs, altering both their nuclear localization and activity, thereby exacerbating impairment of thyroid retinoid receptor-mediated transcription (18).

Interrelationships Between Thyroid Hormones and Retinoid Pathways

Multivariate regression analysis revealed that FT3 and FT4 independently predicted retinol levels, while RBP was strongly influenced by retinol, cholesterol metabolism, and inversely associated with LDL. These findings illustrate the close metabolic interdependence between thyroid hormones and retinoid homeostasis, consistent with the established role of thyroid hormones in regulating hepatic lipid and protein synthesis (19). They also highlight the sensitivity of RBP to lipid metabolism, given that it circulates predominantly in complex with transthyretin a protein whose synthesis is influenced by thyroid status. These interrelationships raise the possibility of a feedback mechanism wherein impaired retinoid signalling may hinder T4 to T3 conversion or nuclear receptor activation, potentially contributing to suboptimal responsiveness to LT_4 therapy.

Clinical Implications

Overall, the findings indicate that despite LT_4 treatment, significant disturbances persist in Vitamin A transport, nuclear receptor expression, and downstream intracellular signalling pathways. Such disruptions may partially account for the persistent symptoms reported by some hypothyroid patients despite normalization of TSH levels. The evaluation of retinoid markers particularly RBP may therefore be clinically useful in identifying patients with impaired tissue level thyroid hormone action. Furthermore, the observed perturbations in the RXR/RAR – TR signalling axis and increased MAPK activity suggest that targeted micronutrient supplementation or interventions aimed at enhancing retinoid signalling may improve therapeutic outcomes, though such strategies require validation in future interventional studies (20).

Strengths and Limitations

The strengths of this study include the integration of biochemical and molecular markers, stratification by autoimmune status and disease severity, and the application of multivariate

modelling to identify independent determinants of retinoid dysfunction. However, several limitations must be acknowledged. The cross-sectional design precludes causal inference, PBMC gene-expression patterns may not fully reflect thyroid tissue physiology, and dietary Vitamin A intake was not assessed. Future prospective and mechanistic studies are needed to clarify the causal pathways linking thyroid dysfunction and retinoid disturbances and to determine whether correcting Vitamin A impairment enhances LT_4 treatment responsiveness.

7. Conclusion

Hypothyroidism impairs Vitamin A functional status by reducing both retinol and RBP levels, with RBP emerging as a more sensitive biomarker that better distinguishes disease severity and detects subtle metabolic disturbances. These findings highlight the importance of including retinoid status in the clinical evaluation of patients with hypothyroidism.

8. Summary

Hypothyroid patients exhibit significant disturbances in retinoid markers such as retinol and RBP, along with marked alterations in molecular pathways including RXR, RAR, TRα, ERK, and p38 MAPK. Among them, individuals with Hashimoto's hypothyroidism show the most pronounced impairments. RBP emerges as a more sensitive biomarker than retinol for identifying vitamin A dysregulation in these patients. Moreover, strong correlations are observed between thyroid hormone levels and key components of the retinoid pathway, and the associated gene-expression profiles indicate disrupted nuclear-receptor signalling coupled with increased activation of cellular stress pathways.

9. References:

1. Usha Menon V, Sundaram KR, Unnikrishnan AG, Jayakumar RV, Nair V, Kumar H. High prevalence of undetected thyroid disorders in an iodine sufficient adult south Indian population. J Indian Med Assoc. 2009 Feb;107(2):72–7.
2. Ragusa F, Fallahi P, Elia G, Gonnella D, Paparo SR, Giusti C, et al. Hashimotos' thyroiditis: Epidemiology, pathogenesis, clinic and therapy. Best Pract Res Clin Endocrinol Metab. 2019 Dec;33(6):101367.
3. Chaker L, Bianco AC, Jonklaas J, Peeters RP. Hypothyroidism. Lancet Lond Engl [Internet]. 2017 Sept 23 [cited 2025 Dec 7];390(10101):1550–62. Available from: https://pmc.ncbi.nlm.nih.gov/articles/PMC6619426/
4. Perros P, Van Der Feltz-Cornelis C, Papini E, Nagy EV, Weetman AP, Hegedüs L. The enigma of persistent symptoms in hypothyroid patients treated with levothyroxine: A narrative review. Clin Endocrinol (Oxf). 2023 Apr;98(4):461–8.
5. Capriello S, Stramazzo I, Bagaglini MF, Brusca N, Virili C, Centanni M. The relationship between thyroid disorders and vitamin A.: A narrative minireview. Front Endocrinol [Internet]. 2022 Oct 11 [cited 2025 Dec 7];13:968215. Available from: https://pmc.ncbi.nlm.nih.gov/articles/PMC9592814/

6. Suzuki M, Tomita M. Genetic Variations of Vitamin A-Absorption and Storage-Related Genes, and Their Potential Contribution to Vitamin A Deficiency Risks Among Different Ethnic Groups. Front Nutr [Internet]. 2022 Apr 28 [cited 2025 Dec 7];9. Available from: https://www.frontiersin.org/journals/nutrition/articles/10.3389/fnut.2022.861619/full
7. Jonklaas J, Bianco AC, Bauer AJ, Burman KD, Cappola AR, Celi FS, et al. Guidelines for the Treatment of Hypothyroidism: Prepared by the American Thyroid Association Task Force on Thyroid Hormone Replacement. Thyroid [Internet]. 2014 Dec 1 [cited 2025 Dec 7];24(12):1670–751. Available from: https://pmc.ncbi.nlm.nih.gov/articles/PMC4267409/
8. Newcomer ME, Ong DE. Retinol Binding Protein and Its Interaction with Transthyretin. In: Madame Curie Bioscience Database [Internet] [Internet]. Landes Bioscience; 2013 [cited 2025 Dec 7]. Available from: https://www.ncbi.nlm.nih.gov/books/NBK6223/
9. Capriello S, Stramazzo I, Bagaglini MF, Brusca N, Virili C, Centanni M. The relationship between thyroid disorders and vitamin A.: A narrative minireview. Front Endocrinol. 2022;13:968215.
10. Xiao S, Jin H, Korn T, Liu SM, Oukka M, Lim B, et al. Retinoic acid increases Foxp3+ regulatory T cells and inhibits development of Th17 cells by enhancing TGF-β-driven Smad3 signaling and inhibiting IL-6 and IL-23 receptor expression. J Immunol Baltim Md 1950 [Internet]. 2008 Aug 15 [cited 2025 Dec 7];181(4):2277–84. Available from: https://pmc.ncbi.nlm.nih.gov/articles/PMC2722959/
11. Chen X, Huang F, Qi Y, Zhou M, Yin Q, Peng Y, et al. Serum and thyroid tissue level of let-7b and their correlation with TRAb in Graves' disease. J Transl Med. 2018 July 5;16(1):188.
12. McAninch EA, Rajan KB, Miller CH, Bianco AC. Systemic Thyroid Hormone Status During Levothyroxine Therapy in Hypothyroidism: A Systematic Review and Meta-Analysis. J Clin Endocrinol Metab [Internet]. 2018 Aug 15 [cited 2025 Dec 7];103(12):4533–42. Available from: https://pmc.ncbi.nlm.nih.gov/articles/PMC6226605/
13. Thyroid Hormone Regulation of Metabolism | Physiological Reviews | American Physiological Society [Internet]. [cited 2025 Dec 7]. Available from: https://journals.physiology.org/doi/full/10.1152/physrev.00030.2013?utm_source=chatgpt.com
14. Centanni M, Maiani G, Parkes AB, Ferro Luzzi A, Lazarus JH. THYROID HOMEOSTASIS AND RETINOL CIRCULATING COMPLEX RELATIONSHIPS IN A SEVERE IODINE-DEFICIENT AREA OF SENEGAL. 1995 [cited 2025 Dec 7]; Available from: https://iris.uniroma1.it/handle/11573/101308?utm_source=chatgpt.com
15. Sombié OO, Zeba AN, Somé JW, Kazienga A, Bationo JF, Davis C, et al. Association between vitamin A status, inflammations, and infections in children 36-59 months of age in rural Burkina Faso: A cross - sectional study. J Health Popul Nutr. 2025 Apr 5;44(1):107.
16. Rubin LP, Ross AC, Stephensen CB, Bohn T, Tanumihardjo SA. Metabolic Effects of Inflammation on Vitamin A and Carotenoids in Humans and Animal Models. Adv Nutr [Internet]. 2017 Mar 1 [cited 2025 Dec 7];8(2):197–212. Available from: https://www.sciencedirect.com/science/article/pii/S2161831322007116
17. Stoney PN, Helfer G, Rodrigues D, Morgan PJ, McCaffery P. Thyroid hormone activation of retinoic acid synthesis in hypothalamic tanycytes. Glia [Internet]. 2016 Mar [cited 2025 Dec 7];64(3):425–39. Available from: https://pmc.ncbi.nlm.nih.gov/articles/PMC4949630/
18. Li D, Li T, Wang F, Tian H, Samuels HH. Functional evidence for retinoid X receptor (RXR) as a nonsilent partner in the thyroid hormone receptor/RXR heterodimer. Mol Cell Biol. 2002 Aug;22(16):5782–92.
19. Lee S, Privalsky ML. Heterodimers of Retinoic Acid Receptors and Thyroid Hormone Receptors Display Unique Combinatorial Regulatory Properties. Mol Endocrinol [Internet]. 2005 Apr 1 [cited 2025 Dec 7];19(4):863–78. Available from: https://doi.org/10.1210/me.2004-0210
20. Hidalgo J, Vallejo BA, Soto Jacome C, Ayala IN, Cordova S, Duran M, et al. Real Practice Assessment of Persistent Symptoms After Initiation of Levothyroxine. Endocr Pract [Internet]. 2024 Feb [cited 2025 Dec 7];30(2):95–100. Available from: https://www.scopus.com/pages/publications/85178385607

Annexure I (Publications)

1 Viswanathan I, Vinayagam V. A comprehensive review of the beneficial and adverse effects of levothyroxine therapy in health and disease. Zhuzao/Foundry [Internet]. 2025 Jan 31 [cited 2025 Dec 8];28(1):62-68. Available from: doi:10.29014/FJ-2025-1001-4977.89.1983
2 Viswanathan I, Santhoshakumari TMJ, Vinayagam V, Green SR. Impact of physical exercise on biochemical and hematological indices in hypothyroidism: integration of a patient-centered activity questionnaire with levothyroxine therapy. Zhuzao/Foundry [Internet]. 2025 Sep 27 [cited 2025 Dec 8];28(9):97-109. Available from: doi:10.29014/FJ-2025-1001-4977.89.2171
3 Viswanathan I, Vinayagam V, Muraliswaran P. Biochemical signatures of hypothyroidism: comparative and correlation analyses in overt and subclinical subtype. Int J Environ Sci [Internet]. 2025 Sep 29 [cited 2025 Dec 8];11(24s). Available from: https://doi.org/10.64252/jm8wmr55
4 Viswanathan I, Vinayagam V, Santhoshakumari TMJ. Enhancing clinical chemistry laboratory performance: analyzing the impact of intermittent training and feedback on quality indicators in tertiary care settings. Zhuzao/Foundry [Internet]. 2025 Mar 5 [cited 2025 Dec 8];28(3):10-19. Available from: doi:10.29014/FJ-2025-1001-4977.89.1992
5 Viswanathan I, Santhoshakumari TMJ, Vinayagam V, Green SR. Assessment of anti-TPO and anti-TG antibody levels in autoimmune thyroid disease among levothyroxine-treated hypothyroid patients. J Rare Cardiovasc Dis. 2025 Dec 6 [cited 2025 Dec 8]; 25-S6:-. doi:10.61336/jrcd/25-S6-117.

OTHER PUBLICATIONS:

6 Vinayagam V, Keerthana S, Viswanathan I, Vinodbabu S, Santhoshakumari TMJ, Sumathi S. Evaluation of calculated HbA1C as a low–cost alternative for management of diabetes mellitus for possible use in low–resource settings. J Chem Health Ris [Internet]. 2025 Mar 20 [cited 2025 Dec 8];15(2):324-328. ISSN: 2251-6727.
7 Vinayagam V, Naganna RR, Ghose S, Viswanathan I, Karthikeyan K, Saravanakumar S, et al. Serum sex hormone binding globulin (SHBG) as an accessory diagnostic marker in hypertensive disorders of pregnancy: a cross sectional study in a tertiary care centre. Zhuzao/Foundry [Internet]. 2025 Feb [cited 2025 Dec 8];28(2):79-83. Available from: doi:10.29014/FJ-2025-1001-4977.89.1989

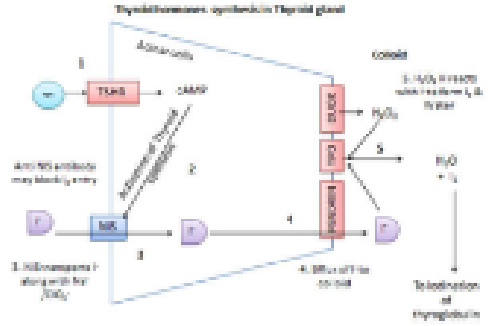
A Comprehensive Review of the Beneficial and Adverse Effects of Levothyroxine Therapy in Health and Disease

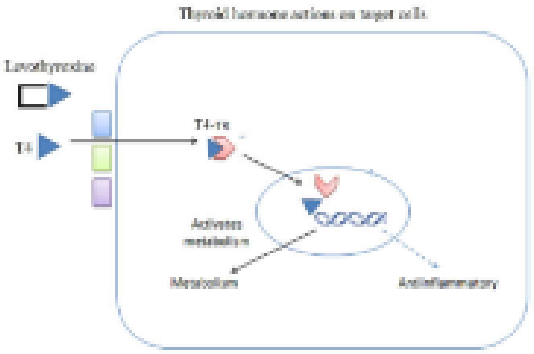
Induja Viswanathan[1], Vickneshwaran Vinayagam[1],

Affiliation- Department of Biochemistry, Mahatma Gandhi Medical College and Research Institute, Sri Balaji Vidyapeeth (Deemed-to-be university), Puducherry, India

Abstract:

Levothyroxine is a synthetic form of the thyroid hormone used to treat hypothyroidism, a condition where the thyroid gland does not produce enough hormones. It helps normalize thyroid hormone levels, improving symptoms such as fatigue, weight gain, cold intolerance, and cognitive difficulties. Levothyroxine also supports metabolism, heart function, and mood. However, its use can lead to side effects, particularly if the dose is not properly adjusted. Common side effects of an overdose include increased heart rate, anxiety, and weight loss, while an underdose can result in fatigue, weight gain, and depression. Long-term use of high doses may also affect bone health and increase the risk of osteoporosis. Regular monitoring of thyroid hormone levels is essential to ensure the correct dosage, and interactions with other medications should be considered. Levothyroxine is generally safe when used appropriately but requires careful management to avoid complications.





Keywords: Levothyroxine, hypothyroidism, thyroid hormone, thyroid treatment, medication management.

Impact of Physical Exercise on Biochemical and Hematological Indices in Hypothyroidism: Integration of a Patient - Centered Activity Questionnaire with Levothyroxine Therapy

Induja Viswanathan[1], TMJ Santhoshakumari[2], Vickneshwaran Vinayagam[3*], Siva Ranganathan Green[4],

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2.Professor, Department of Biochemistry, Mahatma Gandhi Medical College and Research Institute, Sri Balaji Vidyapeeth (Deemed-to-be-University), Puducherry, India.

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*Corresponding Author: Dr. Vickneshwaran Vinayagam, PhD, Department of Biochemistry, Mahatma Gandhi Medical College and Research Institute, Sri Balaji Vidyapeeth (Deemed-to-be-University), Puducherry, India.

Abstract

Background:

Hypothyroidism is associated with persistent metabolic and hematological disturbances, even during levothyroxine therapy. Physical exercise improves metabolism and immune function, but its impact in hypothyroid patients is underexplored.

Objective:

To evaluate the effect of physical exercise on biochemical and hematological indices in hypothyroid patients receiving levothyroxine therapy, using a patient-centered activity questionnaire.

Methods:

A cross-sectional study included 80 hypothyroid patients on stable levothyroxine and 60 age- and sex-matched controls. Physical activity was assessed using a validated questionnaire. Biochemical markers (FT3, FT4, TSH, lipid profile, HbA1c) and hematological indices (hemoglobin, RBC, hematocrit, WBC, platelets) were measured and correlated with activity levels.

Journal of Rare Cardiovascular Diseases

ISSN: 2299-3711 (Print) e-ISSN: 2300-5505 (Onlir

Assessment of Anti-TPO and Anti-TG Antibody Levels in Autoimmune Thyroid Disease Among Levothyroxine-Treated Hypothyroid Patients

Induja Viswanathan [1], *TMJ Santhoshakumari* [1], *Vickneshwaran Vinayagam* [1], *Siva Ranganathan Green,* [2]

DOI: https://doi.org/10.61336/jrcd/25-S6-117

Show Less

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[2] Department of Medicine, Mahatma Gandhi Medical College and Research Institute, Sri Balaji Vidyapeeth (Deemed-to-Be-University), India

Received: 2025-10-08 Revised: 2025-10-29 Accepted: 2025-11-20 Published: 2025-12-06

Full Text

PDF

Abstract

Objectives To assess anti-thyroid peroxidase (anti-TPO) and anti-thyroglobulin (anti-TG) antibody levels in levothyroxine (LT_4)–treated primary hypothyroid patients and evaluate their associations with thyroid hormone profile and disease duration. *Materials and Methods* A case–control analytical study was conducted among 80 LT_4-treated primary hypothyroid patients and 60 age- and sex-matched euthyroid controls. Thyroid function tests (TSH, fT3, fT4) were measured using chemiluminescence immunoassay, while anti-TPO and anti-TG antibodies were assessed using ELISA. *Statistical Analysis* Normality was evaluated using the Shapiro–Wilk test. Between-group comparisons were performed using the independent t-test or Mann–Whitney U test. Correlations between antibody titers, thyroid hormone levels, and disease duration were assessed using Pearson's or Spearman's correlation coefficients. A p-value <0.05 was considered statistically significant. *Results* Anti-TPO and anti-TG positivity were significantly higher in LT_4-treated patients compared with controls (65% vs 3% and 47.5% vs 2%, respectively; p <0.001). Treated patients showed elevated TSH and reduced fT3 and fT4 (p <0.001). Anti-TPO correlated positively with TSH (r = +0.48) and negatively with fT4 (r = –0.32), while anti-TG correlated positively with disease duration (r = +0.41). Dual-antibody positivity showed the strongest association with elevated TSH (r = +0.52). *Conclusions* Persistent elevation of anti-TPO and anti-TG antibodies in LT_4-treated hypothyroid patients indicates ongoing autoimmune thyroid damage despite hormone replacement. Regular monitoring of thyroid autoantibodies alongside biochemical markers may improve risk stratification, guide LT_4 dose optimization, and enhance long-term management of autoimmune hypothyroidism.

Keywords

Autoimmune thyroiditis; Anti-TPO; Anti-TG; Levothyroxine therapy; Hypothyroidism; Thyroid autoantibodies.

ORIGINAL ARTICLE

Enhancing Clinical Chemistry Laboratory Performance: Analyzing the Impact of Intermittent Training and Feedback on Quality Indicators in Tertiary Care Settings.

Induja Viswanathan[1], Vickneshwaran Vinayagam[1*], TMJ Santhoshakumari[1]

1. Department of Biochemistry, Mahatma Gandhi Medical College and Research Institute, Sri Balaji Vidyapeeth (Deemed-to-be university), Puducherry, India

Corresponding Author: Vickneshwaran Vinayagam, Department of Biochemistry, Mahatma Gandhi Medical College and Research Institute, Sri Balaji Vidyapeeth (Deemed-to-be university), Puducherry, India, E-mail id: vickneshwaranvinayagam@mgmcri.ac.in

ABSTRACT:

Objectives: The objective of the study is to evaluate and improve the quality indicators with respect to the manual errors in total testing process in a clinical chemistry laboratory investigation by giving suitable intervention to the laboratory technical staff.

Materials and Methods: The Quality indicators of pre-analytical, analytical and post-analytical phases were documented for a period of 4 months and sensitization given for lab personnel for a period of 1 month regarding the laboratory errors that frequently occurred during the study period. Their knowledge was assessed by conducting pre-test and post-test, by means of multiple choice questions. After sensitization the Quality indicators of pre-analytical, analytical and post-analytical phases were documented for a further period of 4 months. Laboratory errors of the three phases were compared before and after sensitization and were expressed as percentage.

Statistical analysis: Descriptive statistics were used to assess the categorical data using SPSS statistical software version 19.0 and the values are represented as frequency (n) and percentage distribution of relative frequency (%).

Annexure II (IHEC certificate)

SRI BALAJI VIDYAPEETH
DEEMED TO BE UNIVERSITY

MAHATMA GANDHI MEDICAL COLLEGE & RESEARCH INSTITUTE

INSTITUTIONAL HUMAN ETHICS COMMITTEE

CDSCO – ECR /451/ Inst/ PO/ 2013/ RR – 19 DHR - EC/NEW/INST/2022/PY/0169

MEMBERS PRESENT DURING THE MEETING

CHAIRPERSON
DR. P. JAMBULINGAM
Chair (Vector Biology),
Former Director,
ICMR-Vector Control Research Centre,
Department of Health Research,
MH & FW, Puducherry.

MEMBERS
DR. MOHAMED HANIFAH
Professor & Head – CCU
Dept. of Medicine,
MGMCRI, Puducherry.
Dr. KRISHNAGOPAL R
Professor & Head,
Dept. of Orthopedics
MGMCRI, Puducherry
DR. MOHANDOSS
Advocate
Labour and Service laws Consultant
Puducherry.
MRS. PRABHAVATHI
Assistant professor
Sri Saradha College of Education
Villupuram
DR. KARTIKEYAN K
Associate Professor
Dept. of Paediatrics,
MGMCRI, Puducherry
DR. UMA NARAYANAMURTHY
Associate Professor
Dept. of Pharmacology
MGMCRI, Puducherry.
Dr. INDHIRA S
Professor & Head
Dept. of Philosophy
Pondicherry university, Puducherry
Dr. SHAHIN SULTANA
Professor of Social Worker
Pondicherry university, Puducherry
MEMBER SECRETARY
DR. BARATHANE D
Associate Professor
Dept. of Pharmacology,
MGMCRI, Puducherry.
Email: ihec@mgmcri.ac.in

CERTIFICATE OF APPROVAL

Project No: MGMCRI/2023/02/IHEC/106 **Date:29/12/2023**

Project Title:

IMPACT OF GENETIC EXPRESSION OF VITAMIN A FUNCTIONAL STATUS IN LEVOTHYROXINE TREATED HYPOTHYROIDISM

Study period: This approval is valid for the **one year** duration of the study and shall be considered for extension by IHEC, upon request with in sufficient time by the PI.

Principal Investigator: Ms. V. INDUJA

Type of review:
Full Board Review

Date of review (D/M/Y): 04/12/2023
Date of previous review, if revised application:

- ✓ It is understood that the study will be conducted under your direction, in a total of **120** research participants, at as per the submitted protocol.
- ✓ **The IHEC approves the above mentioned study**
- ✓ For studies which will continue for more than a year, a continuing review report needs to be submitted (within1 month of the due date i.e. 11 months from the date of approval) on or before **29/11/2024**

29/12/23

Dr. BARATHANE D
MEMBER SECRETARY
Institutional Human Ethics Committee
MGMCRI, Puducherry - 607 402

ANNEXURE III (PRE PhD RESULT CERTIFICATE)

SRI BALAJI VIDYAPEETH

DEEMED TO BE UNIVERSITY
DECLARED U/S 3 OF THE UGC ACT, 1956

ACCREDITED BY NAAC
WITH 'A++' GRADE

PILLAIYARKUPPAM, PONDICHERRY - 607 402.

OFFICE OF THE CONTROLLER OF EXAMINATIONS
Dr. **S. SRIRANGARAJ**
Controller of Examinations

Date: 23-07-2024

No. SBV/COE/Ph.D-Result/May-2024/A5

UNIVERSITY MEMORANDUM

Sub : Sri Balaji Vidyapeeth – Ph.D Degree in Part – I Course Work Assessment – Results Notified.

* * * * *

Mr/Mrs/Dr. **INDUJA V,** UIN. **2205003001** Research Scholar in **MEDICINE (Full Time)** is declared to have passed the Ph.D Part-I – Course Work Assessment conducted in May - 2024 and he/she is permitted to continue the research under the guidance of **Dr. MURALISWARAN P** and submit the thesis for the award of Ph.D Degree as per the regulations of SBV.

Sl. No.	Subjects	Credits	Marks (%)	Result Status
1	PAPER - I RESEARCH METHODOLOGY AND BIOSTATISTICS	4	78 %	PASS
2	PAPER - II ON CHOSEN FIELD OF RESEARCH	6	81 %	PASS

S. Sriranga raj

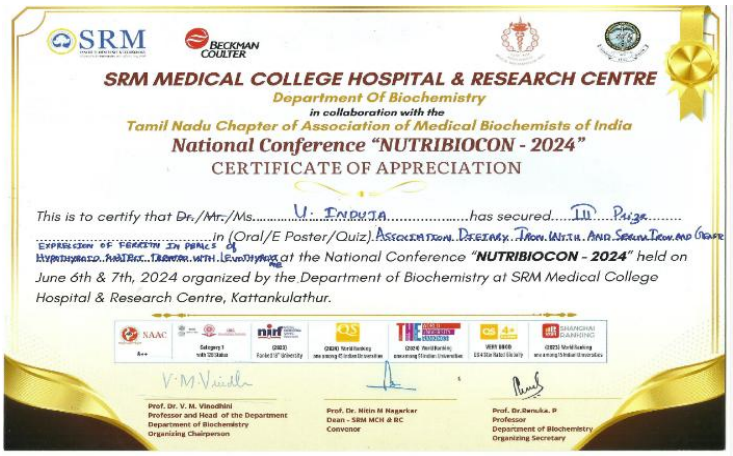
CONTROLLER OF EXAMINATIONS

To : Mr/Mrs/Dr. **INDUJA V** UIN. **2205003001**

ANNEXURE IV (SCIENTIFIC PRESENTATIONS CERTIFICATE)

3RD ANNUAL CONGRESS OF ARAB THYROID ASSOCIATION
CERTIFICATE OF ABSTRACT ORAL PRESENTATION
THIS IS TO CERTIFY THAT
WINNER
INDUJA VISHWANATHAN
HAS DELIVERED THEIR ABSTRACT PRESENTED AS
ORAL PRESENTATION DURING THE SCIENTIFIC ACTIVITIES OF THE
3RD ANNUAL ARAB THYROID ASSOCIATION CONGRESS
AND AWARDED "II WINNER" FOR ORAL PRESENTATION
Dr. Nasser Aljuhani
Dr. Eiman Alseddeeqi
Dr. Wiam Ibrahim Hussein
Dr. Thamer Alessa

CERTIFICATE
OF ABSTRACT SUBMISSION
This is to certify that
INDUJA VISWANATHAN
has delivered an abstract as an oral presentation titled
VEGF & Neurobiomarkers in Hypothyroidism
during the scientific activities of the
4th Arab Thyroid Congress (ArTA 2025)
held on
May 23-24, 2025, at Holiday Inn Dubai Festival City, UAE


SRM
BECKMAN COULTER
SRM MEDICAL COLLEGE HOSPITAL & RESEARCH CENTRE
Department Of Biochemistry
in collaboration with the
Tamil Nadu Chapter of Association of Medical Biochemists of India
National Conference "NUTRIBIOCON - 2024"
CERTIFICATE OF APPRECIATION
This is to certify that Dr./Mr./Ms. U. INDUJA has secured III Prize
in (Oral/E Poster/Quiz). ASSOCIATION DIETARY IRON WITH SERUM IRON AND GENE EXPRESSION OF FERRITIN IN PBMCS OF HYPOTHYROID PREG. TREATED WITH LEVOTHYROXINE
at the National Conference "NUTRIBIOCON - 2024" held on
June 6th & 7th, 2024 organized by the Department of Biochemistry at SRM Medical College Hospital & Research Centre, Kattankulathur.
Prof. Dr. V. M. Vinodhini
Professor and Head of the Department
Department of Biochemistry
Organizing Chairperson
Prof. Dr. Nitin M Nagarkar
Dean - SRM MCH & RC
Convenor
Prof. Dr.Renuka. P
Professor
Department of Biochemistry
Organizing Secretary

PSGR
Krishnammal College for Women
KCW
CERTIFICATE
This is to certify that
Dr./Mr./Mrs./Ms. V. INDUJA, MAHATMA GANDHI MEDICAL COLLEGE & RESEARCH INSTITUTE has Presented / Participated (ORAL)
online on ASSOCIATION DIETARY IRON WITH SERUM IRON TREATED WITH LEVOTHYROXINE in
the "DST SERB" sponsored "TWO-DAY INTERNATIONAL CONCLAVE ON ONE HEALTH" organized by the
Centre of Excellence in Life Sciences, PSGR Krishnammal College for Women, Coimbatore, India on 18
& 19, September 2024.
Dr. C. Krishnaveni
CONVENOR
Dr. R. Nirmal Kumar
ORGANIZING SECRETARY
Dr. P.B. Harathi
PRINCIPAL

MGMARI
Department of Health Research
Ministry of Health and Family Welfare
Government of India
MAHATMA GANDHI MEDICAL ADVANCED RESEARCH INSTITUTE
SRI BALAJI VIDYAPEETH
(DEEMED TO BE UNIVERSITY)
Puducherry, India
Certificate of Participation
This is to certify that Mr./ Mrs./ Dr. Induja Viswanathan from MGMCRI, Sri Balaji Vidyapeeth
has presented Oral Titled "Altered expression of stress-sensitive signalling in PBMCs of hypothyroid subjects treated with levothyroxine" in
" National Symposium and Workshop on Zebrafish Genome
Editing Knockout Technology and Research Applications"
09th & 10th October 2025
Sponsored by : Department of Health Research (DHR), Government of India
Organized by : Mahatma Gandhi Medical Advanced Research Institute (MGMARI)
Prof. Nhiar Ranjan Biswas
Dr. K.S. Reddy
Dr. Parkash Babu Narasimhan
Dr. Rakesh Sarma M
Dr. Agri Ganeche
Dr. Raghavender Medishetti

TAGORE MEDICAL COLLEGE & HOSPITAL
(Affiliated to the Tamilnadu DR.M.G.R Medical University)
Vandalur-Kelambakkam Road,Rathinamangalam,Chennai-600127
NAAC
Certificate Of Participation
This Certificate is Presented To
V Induja
For participating in the E-POSTER Competition conducted in the National Conference titled
"BIOCHEM-INFERTILITY NEXUS 2024" on September 20th & 21st,2024, by the Department of
Biochemistry,Tagore Medical College & Hospital, Chennai
Topic : Association Dietary Iron With Serum Iron And Genetic Expression Of Ho - 1 In Pbmcs Of Hypothyroidism Pregnancy Treated With Levothyroxine
Dr.S.Shenbaga Lalitha Organizing Chairperson
Dr.J.Muthukumaran Dean
Dr.D.H.Gopalan Vice principal